morphine significantly elevated response thresholds ($P<0.01$ for central; $P<0.001$ for medial, related t-tests). In medial, but not central, sites naloxone antagonized the morphine effect; naloxone alone had no significant effect at either site.

Exploration was measured in a holeboard (4 equally spaced holes in a floor 65 x 65 cm). Infrared photocells connected to counters provided automated measures of exploration (the number of head dips and the time spent head-dipping) and locomotor activity (File & Wardill, 1975). Rats received a single 10 min trial and were tested in randomized order between 0800–1200 h. When injected into the medial amygdaloid nucleus, morphine significantly reduced duration of head-dipping ($F(1,38) = 5.9$, $P<0.02$), an effect not antagonized by naloxone. However, in the central nucleus, morphine produced naloxone-reversible decreases in both the number of head-dips and time spent head-dipping ($F(1,42) = 6.1$ and 5.1, $P<0.02$ and 0.001, respectively) and in locomotor activity ($F(1,42) = 6.1$, $P<0.02$).

In conclusion, our results suggest that the presence or absence of specific morphine receptors cannot be deduced on the basis of a single behavioural test. We have shown that in the central amygdala morphine acts on specific receptors to reduce exploration and on non-specific receptors to raise electric shock thresholds. The converse appears to apply in the case of the medial amygdala.

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Behavioural effects of chronic amphetamine and their reversal by haloperidol in the marmoset

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Acute administration of amphetamine to the marmoset results in an increase in small head movements (checking), a decrease in activities and social contact, but no change in the amount of locomotion (Ridley, Baker & Crow, 1979; Scraggs & Ridley, 1978a). The increase in checking is blocked by haloperidol although activities and social contact cannot be reinstated by further drug treatment (Scraggs & Ridley, 1978b).

Chronic administration of amphetamine in rodents results in a progressive augmentation of stereotyped behaviour (Segal & Mandell, 1974). In primates, chronic methamphetamine treatment produces a progressive deterioration in purposeful and co-ordinated behaviour (Ellinwood & Kilbey, 1975).

In this study, (+)-amphetamine was administered via the drinking water to an established group of six marmosets in increasing doses of 1–4 mg/kg over 27 days (phase I). Haloperidol (0.01 mg/kg) was then given in addition to amphetamine (4 mg/kg) for 51 days (phase II) followed by 33 days of amphetamine (4 mg/kg) alone (phase III). Finally the animals were observed without drugs for 33 days (phase IV).

Behaviour was observed each day on alternate 3 day periods except when drug treatment changed, when behaviour was observed on the 3 days immediately before and after the change. Means of behaviour over 3 day periods were calculated and compared with pre-drug control readings (phase 0). On each observation day, each animal was observed for 100 s. Behaviour was classified each second into 5 mutually exclusive categories:

1. Checking: movement of the head only.
2. Activities: movement of only part of the body excluding head only. This category included activities such as eating, drinking and self-grooming.
3. Social contact: physical contact between 2 animals including grooming, fighting, play and sexual activity.
4. Locomotion: displacement of the whole body.
5. Inactivity: no discernible movement.

It was found that during phase I checking increased initially ($P<.001$) but then declined to normal levels: locomotion was initially unchanged but then declined ($P<.05$). Social contact was reduced ($P<.05$) while inactivity was increased ($P<.01$) throughout phase I. Purposeful activities were initially slightly decreased, but not significantly. However, during phase I a syn-

drome of destructive self-grooming emerged.

When haloperidol was administered (phase II) destructive self-grooming ceased abruptly; checking and social contact were unaltered while inactivity was increased further ($P<.001$). Locomotion was initially further decreased ($P<.01$) but returned to normal levels by the end of phase II.

When haloperidol was withdrawn (phase III) checking was initially increased ($P<.001$) but then returned to normal (cf phase I). Destructive self-grooming did not reappear. Locomotion and social contact remained unchanged while inactivity was comparable to that during phase I.

During phase IV when no drugs were administered, all behaviours including social contact were within the normal range except inactivity which was elevated initially ($P<.05$).

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Effects of spiperone on feeding parameters in the rat and interactions with (+)-amphetamine, mazindol or (±)-fenfluramine

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Spiperone is a neuroleptic with a marked dopamine receptor blocking action (Andén, Butcher, Corrodi, Fuxe & Ungerstedt, 1970), and it has been employed as a dopamine antagonist in feeding experiments (Cooper & Sweeney, 1978; Heffner, Zigmond & Stricker, 1977; Rolls, Rolls, Kelly, Shaw, Wood & Dale, 1974). Previously, we have described a feeding test in which effects of drug treatments are described not only in terms of the amount of food intake, but also in terms of the duration of feeding and the rate at which food is consumed (Cooper & Francis, 1978). This report describes the behavioural effects of spiperone in this test, administered either alone or in combination with the anorectic compounds, (+)-amphetamine, mazindol or (±)-fenfluramine.

The subjects were 144 naive, male, adult black-hooded rats, weighing 220–260 g. On the day before testing, food was removed at 17.00 h, and feeding tests were run the following morning. Each rat was tested for 10 min in a test-cage with familiar food pellets available. The rats were first divided into 3 groups which determined the first injection given i.p. 120 min before the feeding test: (i) tartaric acid (0.1 M, control injection), (ii) spiperone (0.06 mg/kg), (iii) spiperone (0.10 mg/kg). Each group was then subdivided into 6 groups (n = 8/group): (i) mazindol (0.065 mg/kg), (ii) mazindol (1.25 mg/kg), (iii) (+)-amphetamine (0.5 mg/kg), (iv) (+)-amphetamine (1.0 mg/kg), (v) fenfluramine (4.0 mg/kg), (vi) control vehicle injection. These groups determined the second injection given 30 min before the test (mazindrol, s.c.; (+)-amphetamine, (±)-fenfluramine, i.p.).

Spiperone shortened the latency to begin feeding ($F = 10.63$, d.f. 2,63, $P<0.001$), alone and in combination with (+)-amphetamine and mazindol; in contrast, it prolonged the latency in combination with (±)-fenfluramine. Spiperone prolonged the duration of feeding ($F = 11.15$, d.f. 2,63, $P<0.001$), alone and in combination with (+)-amphetamine and mazindol, but at the higher dose (0.10 mg/kg) it significantly reduced the feeding duration in combination with (±)-fenfluramine. As expected, (+)-amphetamine and mazindol markedly prolonged the latency to feed ($F + 10.30$, d.f. 2,63, $P<0.001$; $F = 37.49$, d.f. 2.63, $P<0.001$, respectively), and shortened the duration of feeding ($F = 29.62$, d.f. 2,63, $P<0.001$; $F = 50.50$, d.f. 2,63, $P<0.001$). However, (±)-fenfluramine (4.0 mg/kg) exerted only minor effects on latency and feeding duration. Spiperone markedly reduced the rate of eating ($t = 3.05$, 14 d.f., $P<0.005$), an action it shared with (±)-fenfluramine ($t = 3.23$, 14 d.f., $P<0.005$). Their joint effect on eating rate was additive. In contrast, (+)-amphetamine and mazindol significantly increased eating rate, an effect that was antagonized in the presence of spiperone. All 3 anorectic drugs reduced the amount of food-intake significantly; mazindol and (+)-amphetamine by reducing eating duration but not rate, (±)-fenfluramine by reducing rate but not eating duration. Spiperone also significantly reduced food-intake ($F = 3.46$, d.f. 2,63, $P<0.04$), by reducing eating rate and not by reducing eating duration. Spiperone counteracted the anorectic